REMARKS

Applicant respectfully requests reconsideration. Claims 1-7, 17, 18 and 98-102 were previously pending in this application. Claims 98-100 and 102 are currently withdrawn. No claims have been canceled or added. Claims 3 and 17 have been amended. Claim 3 as amended is dependent on withdrawn claims only and is therefore indicated as being withdrawn. Support for the amendment can be found in the specification at least on page 19, lines 2-3. As a result, claims 1-7, 17, 18 and 101 are pending for examination with claim 1 being an independent claim. No new matter has been added.

Claim Rejections under 35 U.S.C. §112, first paragraph – Enablement

Claims 1-7, 17-18 and 101 are rejected under 35 U.S.C. 112, first paragraph, as allegedly failing to comply with the enablement requirement. According to the Examiner, the specification does not enable one skilled in the art to practice the invention without an undue amount of experimentation. Applicant respectfully traverses. A person of ordinary skill in the art would not have to engage in undue experimentation to practice the claimed invention and the claimed invention was therefore enabled at the time of filing.

The claimed invention pertains to a treatment of an allergic condition other than asthma or eczema by administration of a polymer with a specific charge motif. The specification, together with the art, provides enablement support for the claimed invention. Firstly, the specification provides a genus of well characterized polymers with a specific charge motif. Secondly, the specification shows that polymers as diverse as peptides and polysaccharides show an immunomodulatory effect, as long as the polymers have the recited charge motif (See *e.g.*, Example 1, page 49 and Examples 6 and 7, pages 54-55). Thirdly, the methods of treatment are directed to allergic conditions, which is a single category disease that is well described in the art and is characterized by an increase in serum IgE in the subject. Fourthly, in Examples 5 and 8 of the specification (pages 52-57), Applicant shows that administration of the polymers with the recited charge motif result in a decrease in the level of IgE antibodies. Finally, the teachings of the instant application are supported by the art, which has established the relationship between the specific

charge motif of the polymer and the immunomodulatory effect of the polymer. No more is required to establish enablement of the claimed invention.

The Examiner states in the paragraph bridging pages 5-6 of the Office Action that "the specification does not disclose any examples of PSA1 being used to treat any allergic disease other than asthma" and that "the art is highly unpredictable as to what will be a therapy for any particular disease". However, in contrast to the assertion by the Examiner, the art as to allergic conditions is not unpredictable in so far as it involves the effects of a reduction of IgE levels. The art has established that allergic conditions are characterized by a similar physiological event, namely increased levels of IgE antibodies, and that allergic conditions therefore can be treated by lowering the level of IgE antibodies. An "allergic condition" as defined in the specification on page 12, lines 13-14, is an "acquired hypersensitivity to a substance (allergen)". A hypersensitive reaction against an allergen is characterized by a rapid increase in IgE antibodies against the antigen. In fact, measuring the levels of IgE antibody is a diagnostic tool for determining if a subject is suffering from an allergic condition, and a decrease in serum IgE is correlated with the efficacy of treatment for an allergic condition (See *e.g.*, page 17, lines 28 through page 18, line 1). Thus, a treatment regimen that results in the lowering of allergen specific IgE levels is an effective treatment method for the allergic condition. The application demonstrates that polymers with the recited charge motif result in the decrease of the level of IgE in a model of allergy, that is, an allergic asthma model. An increase in IgE levels is the expected consequence of a challenge with any sensitizing allergen in any model of allergy; it is not a function of the particular model of allergy selected. The observed decrease in IgE levels resulting from treatment with a polymer of the invention is an indication that the polymers of the invention can be used to treat any allergic condition.

The Examiner also states that the specification does not provide adequate support for any isolated polymer among the recited polymers for the claimed methods of treatment. However, in contrast to the assertion by the Examiner, the specification, in combination with the art, establishes the relationship between the structure of the polymers and their physiochemical properties. In Examples 5 and 8 of the specification (pages 52-57), Applicant shows that administration of the polymers with the recited charge motif results in a decrease in the level of IgE antibodies. In addition, the specification provides that polymers as diverse as peptides and polysaccharides have a

consistent immunomodulatory effect, as long as the polymers have the recited charge motif (See *e.g.*, Example 1, page 49 and Examples 6 and 7, pages 54-55). Furthermore, the specification has incorporated by reference US 5,679,654, US 5,700,787 and WO 00/59515. These documents provide a detailed analysis of polymers with specific charge motifs and their immunomodulatory effects. The teachings in these documents show that polymers with the motif recited in the instant application have a predictable and consistent immunomodulatory effect, while polymers with a slightly different charge motif do not have such an effect (See *e.g.*, WO 00/59515, pages 35-43). Thus, a person of ordinary skill in the art can, without undue experimentation, determine if a polymer can be used to treat an allergic condition according to the methods of the claimed invention, namely by determining if the polymer has the recited charge motif.

The Examiner refers to Kalka-Moll et al. to support the assertion that the description encompasses many species that the art shows would not work in the claimed invention. Respectfully, the Examiner has misinterpreted the teachings of Kalka-Moll et al. The cited reference evaluates the relationship between length and concentration of the administered polymer and its immunomodulatory effect and teaches that the *potency* of the immunomodulatory effect is dependent on the concentration and the length of the administered polymer. Thus, a person of ordinary skill in the art can rely on the teachings of Kalka-Moll et al. to determine which concentration and length of polymer is required to obtain an optimal immunomodulatory effect. The teachings of Kalka-Moll et al. therefore further support the enablement of the claimed invention.

The Examiner also states that the examples encompass treating asthma include PSA1 and CP1, but that support for the terms "polysaccharide" or "capsular polysaccharide" in a method of treating an allergic condition other than asthma is not adequately disclosed. However, in contrast to the assertion by the Examiner, the specification provides enablement support for the use of polysaccharides and capsular polysaccharides as long as they comprise the recited charge motif, in the treatment of asthma and allergic conditions other than asthma. In addition, the specification also provides support for the treatment of asthma and allergic conditions other than asthma using any polymer with the recited charge motif. As acknowledged by the Examiner, the specification provides examples for the treatment of asthma with PSA1 and CP1. The examples demonstrate

that the levels of anti-allergen IgE antibodies are lowered upon administration of the polymers. As the art provides that the lowering of IgE levels is a general treatment method for all allergic conditions, the examples support the enablement of the treatment off asthma and allergic conditions other than asthma. Furthermore, the teachings of the specification show that PSA1 and CP1 have an immunomodulatory effect and the specification provides which polymers, in addition to PSA1 and CP1 show an immunomodulatory effect. Namely, any polymer with the recited charge motif. Thus, these teachings combined provide that polymers with the recited charge motif enable the treatment of an allergic condition other then asthma.

The Examiner also reasons that the term "comprising" is open language, and that the scope of the claim therefore includes the addition of additional molecules, or the possibility that the methods for treating asthma are not the result of the charge motif of the polymers. Further, according to the Examiner, a person of ordinary skill in the art would not know what can be added to the recited polymer that would not impact the ability of the polymer to treat the allergic disease. Applicant respectfully questions the relevance of the Examiner's argument. Applicant has shown that the polymers with the recited charge motif can be used to treat allergic conditions. The hypothesis by the Examiner that additional molecules may negatively or positively impact the treatment of allergic conditions is not a legal basis for questioning the enablement of the claimed invention.

Further, according to the Examiner, the genus of polymers encompassed by the instant recitation is limitless. Applicant respectfully notes that the genus is circumscribed by a precise definition of a particular charge motif, separating the genus from all other polymers. Applicants have shown that polymers with such a charge motif can be used to treat allergic condition. As the class is clearly defined and the claimed functionality has been shown to be responsible for the activity of the polymers, the requirement for enablement has been met.

Also, according to the Examiner, the claims are not enabled for treating a patient who is free of "symptoms otherwise calling for treatment with the polymer". Applicant respectfully disagrees with this assertion. The specification (page 13, lines 17-26) provides examples of subjects that are free of indications that would otherwise call for treatment with the polymer. These subjects include subjects that do not have an infection, surgery, trauma, a Th1 cell responsive disorder etc. Thus, a

person of ordinary skill in the art would not be required to engage in undue experimentation to determine if a subject is free of "symptoms otherwise calling for treatment with the polymer".

Further, according to the Examiner, the term "anti-IgE" is not enabled. Applicant respectfully disagrees with this assertion. However, without conceding to the Examiner's position and merely in the interest of expediting prosecution, Applicant has amended claim 17 and replaced the term "anti-IgE" with "anti-IgE antibodies", as suggested by the Examiner.

Further, according to the Examiner, the recitation of administering comprises delivering an aerosol to an airway of the subject to treat diseases other than asthma is not enabled. The Examiner reasons that it would require an undue amount of experimentation by one of ordinary skill in the art to determine what allergic diseases will be treated by an aerosol delivery mode of administration other than asthma or another respiratory disease. Applicant respectfully disagrees with this assertion. However, without conceding to the Examiner's position and merely in the interest of expediting prosecution, Applicant has amended claim 3 to clarify that only hayfever and allergic rhinitis are treated by an aerosol delivery mode. As acknowledged by the Examiner, the treatment of respiratory diseases, such as hayfever and allergic rhinitis, by an aerosol delivery mode, would not require undue experimentation.

Finally, the Examiner has not established a *prima facie* enablement rejection of the claims that refer to polysaccharide and bacterial capsular polysaccharide polymers as the arguments set forth by the Examiner do not seem to question the enablement of the methods of the rejected claims, wherein the polymer is a polysaccharide or bacterial capsular polysaccharide.

In conclusion, the application provides enablement support for the claimed invention, and the Examiner has not established that a person of ordinary skill in the art would need to engage in undue experimentation to practice the claimed invention.

Accordingly, reconsideration and withdrawal of the rejection is respectfully requested.

Claim Rejections under 35 U.S.C. §112, first paragraph – Written Description

Claims 1-7, 17-18, and 101 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. According to the Examiner, the claims contain subject matter which was not described in the specification in such a way as to reasonably convey to

one skilled in the relevant art that the inventor(s), at the time of filing, had possession of the claimed invention.

Applicant respectfully traverses. Based on the teachings in the specification, a person of ordinary skill in the art would understand that Applicant had possession of the claimed invention at the time of filing of the application.

The rejected claims pertain to a treatment of an allergic condition other than asthma or eczema by administration of a polymer with a specific charge motif. Applicant has shown that correlation exists between the structure of the recited polymers and the function of the claimed methods of treatment. Firstly, the specification provides a genus of well characterized polymers with a specific charge motif. Secondly, the specification shows that polymers as diverse as peptides and polysaccharides show an immunomodulatory effect, as long as they have the recited charge motif (See *e.g.*, Example 1, page 49 and Examples 6 and 7, pages 54-55). Thirdly, the methods of treatment are directed to allergic conditions, which is a single category disease that is well described in the art and is characterized by an increase in serum IgE in the subject. Fourthly, in Examples 5 and 8 of the specification (pages 52-57), Applicant shows that administration of the polymers with the recited charge motif result in a decrease in the level of IgE antibodies. Finally, the findings in the instant application are corroborated in the art, which has established the relationship between specific charge motif of the polymer and the immunomodulatory effect of the polymer. No more is required to show possession of the claimed invention.

The Examiner asserts that the specification does not adequately describe the genus of polymers that can be used in the claimed invention to have the requisite function of treating an allergic condition. As demonstrated above, the genus of polymers is adequately described. The specification provides the charge motif of the polymer that is required to treat an allergic condition (See *e.g.*, page 37, line 2 through page 38, line 10). In addition, Applicant provides a representative number of species of the polymer with the recited motif, including polysaccharides and polypeptides (See *e.g.*, pages 40-44).

The Examiner reasons that the term "comprising" in claim 1 is open language which widens the scope and potentially includes molecules that not have been discovered, and molecules which have the claimed functionality but where the claimed functionality is not due to the recited motif but

because of some other part of the molecule. Respectfully, this argument does not present a legal basis for rejecting the clams. Applicant has shown that the polymers with the recited motif have the claimed effect, which is all that is required to show possession of the claimed invention, regardless of the use of "comprising". That non-discovered polymers may also have the claimed functionality or that other parts of the molecule may exert a similar effect, is not relevant to Applicant's showing of possession of the claimed invention.

The Examiner also asserts that "a patient who is free of symptoms otherwise calling for treatment with the polymer" is not adequately described. However, in contrast to the assertion by the Examiner, the specification (page 13, lines 17-26) provides examples of subjects that are free of indications that would otherwise call for treatment with the polymer. These subjects include subjects that do not have an infection, surgery, trauma, a Th1 cell responsive disorder etc. Thus, a person of ordinary skill in the art would know that Applicant had possession of the term "a patient who is free of symptoms otherwise calling for treatment with the polymer".

Further, according to the Examiner, the term "anti-IgE" is not adequately described. Applicant respectfully disagrees. However, without conceding to the Examiner's position and merely in the interest of expediting prosecution, Applicant has amended claim 17 and replaced the term "anti-IgE" with "anti-IgE antibodies", as suggested by the Examiner under the enablement rejection.

Finally, the Examiner has not established a *prima facie* written description rejection of the claims that refer to polysaccharide and bacterial capsular polysaccharide polymers as the arguments set forth by the Examiner do not seem to question if Applicants had possession of the methods of the rejected claims, wherein the polymer is a polysaccharide or bacterial capsular polysaccharide.

In conclusion, Applicant has shown that correlation exists between the structure of the recited polymers and the function of the claimed methods of treatment and has provided a representative number of species of the recited polymer. Thus, Applicant had possession of the claimed invention at the time of filing of the application.

Accordingly, reconsideration and withdrawal of the rejection is respectfully requested.

Claim Rejections under 35 U.S.C. §102

Claims 1-2, 4-6, and 18 are rejected under 35 U.S.C. §102(e) as allegedly being anticipated by WO 03/075953. According to the Examiner, WO 03/075953 teaches a method of treating acute respiratory distress syndrome, which is an allergic condition other than asthma and eczema.

Applicant respectfully traverses. The Examiner has not met her burden in establishing a *prima facie* anticipation rejection. The Examiner, merely states that acute respiratory distress syndrome is an allergic condition. However, acute respiratory distress syndrome is not an allergic condition, and the Examiner has provided no support for the assertion that acute respiratory distress syndrome is an allergic condition. Applicant has attached the datasheet of the American Lung Association on Acute Respiratory Distress Syndrome (ARDS). The reference classifies ARDS as a sudden failure of the respiratory system. This failure may be caused by lung inflammation and small blood vessel injury due to sepsis, trauma and/or severe pulmonary infection such as pneumonia. ARDS can also be linked to multiple transfusions, inhalation of salt water, smoke inhalation of toxic chemicals, aspiration of vomit, narcotics, sedatives, overdoses of tricyclic antidepressants and shock from any cause. However, there is no link between ARDS and allergy and ARDS is not an allergic condition.

Because WO 03/075953 does not disclose any allergic condition other than asthma and eczema, which are specifically excluded in the rejected claims, WO 03/075953 does not anticipate the rejected claims.

Accordingly, reconsideration and withdrawal of the rejection is respectfully requested.

Claims 1-2, 4-6, and 18 are also rejected under 35 U.S.C. §102(e) as being anticipated by US 2005/0119164. US 2005/0119164 is the U.S. national phase application corresponding to WO 03/075953. For the same reasons as provided above in connection with WO 03/075953, Applicant respectfully requests that the Examiner reconsider and withdraw the rejection of claims 1-2, 4-6, and 18 under 35 U.S.C. §102(e) as being anticipated by US 2005/0119164.

Claim Rejections under 35 U.S.C. §103

Claims 1-7 and 18 are rejected under 35 U.S.C. 103(a) as allegedly being unpatentable over WO 00/59515 in view of Tang et al. (2001) *J. Immunol.* 166:1471-1481. According to the Examiner, WO 00/59515 teaches treating Th1 responsive disorders. Further, according to the Examiner, column 23, line 60 to column 24, line 7 of WO 00/59515 teaches that driving the immune response toward a Th1 response when it is desirable to have a Th1 cytokine response to treat disease, as is the case for allergies.

Applicant notes that WO/59515 does not contain a section "column 23, line 60 to column 24, line 7". Applicant assumes that the Examiner meant "page 31, lines 13-17", as recited under the 103(a) rejection over U.S. Patent 7,026,285.

Applicant respectfully traverses. The combination of the teachings of WO/59515 and Tang et al. does not render obvious the treatment of allergic conditions by administering the isolated polymers of the rejected claims.

Firstly, the Examiner has not met her burden in considering all rebuttal arguments and evidence presented by applicants, as required (MPEP §2145; *Soni*, 54 F.3d at 750, 34 USPQ2d). Applicant presented arguments in the amendment of November 16, 2007. However, the Examiner has not considered those arguments on the merits and merely states that the arguments are unpersuasive.

Secondly, the combination of the teachings of WO00/59515 and Tang et al. does not establish that the isolated polymers of the rejected claims can be used in the treatment of allergic conditions, as the Examiner has misinterpreted the teachings of both WO00/59515 and Tang et al.

Applicant acknowledges the statement by the Examiner that WO00/59515 teaches that the isolated polymers can be used to treat Th1 responsive disorders. However, WO00/59515 does not teach "(d)riving the immune response towards a Th1 response when it is desirable to have a Th1 cytokine response to treat disease, as is the case for allergies". The Examiner refers to page 31, lines 13-17 to support the assertion. The cited paragraph reads "When T cells are stimulated, they can differentiate toward either Th1 or Th2 cytokine production. The invention in this aspect is based on the discovery that the immunomodulating polymers of the invention can activate T cells to meditate cytokine release having a profile of Th1 cytokines and thus useful any time it is desirable to

activate T cells to produce a Th1 cytokine profile." Thus, the reference merely teaches that a Th1 cytokine profile can be induced. WO00/59515 does not teach switching the immune response from a Th2 response to a Th1 response.

Furthermore, the Examiner has misapplied the teachings of Tang et al. to WO00/59515. Tang et al. teaches that the stimulation of the antigen-presenting activity of macrophages to increases Th1 activity. Combining the methods of WO00/59515 with a method for stimulating the antigen-presenting activity of macrophages does not result in the methods of the claimed invention. While, Tang et al. states that an immune switch from a Th2 response to a Th1 response can protect against allergic allergen, a general method for immune switching is not provided.

Thirdly, Tang et al. teaches away from applying its teachings to WO00/59515. WO00/59515 teaches an increase in IL-10 production. Tang et al. teaches that IL-10 is a cytokine necessary for Th2 proliferation, implying that increasing the production of IL-10 would not be favorable when trying to shift form a Th2 to Th1 response. Thus, a person of ordinary skill in the art would be led away from applying the teachings of WO00/59515, disclosing an increase in IL-10 production, to the teachings of Tang et al.

Fourthly, the instant application teaches that allergic conditions can be treated by suppressing the IgE response (See *e.g.*, page 17, lines 28-31 and Example 8 on pages 55-57). While Applicants are not bound by a mechanism, the suppression of IgE can be achieved by suppressing the Th2 response. WO00/59515 teaches that administering the isolated polymers of the rejected claims result in an increase in the production of IL-10. IL-10 is an inducer of the Th2 response. Thus, based on the teachings of WO00/59515, it is not expected that administering the isolated polymers of the rejected claims would result in a suppression of the Th-2 response. Therefore, the instant application provides unexpected results in that the polymers of the rejected claims can suppress IgE levels.

Thus, at least for the reasons presented above, the combination of WO00/59515 and Tang et al. does not render obvious the methods of the rejected claims.

Accordingly, reconsideration and withdrawal of the rejection is respectfully requested.

The Examiner rejected claims 1-7 and 18 under 35 U.S.C. 103(a) as unpatentable over U.S. Patent 7,026,285 in view of Tang et al. (*supra*). Applicant respectfully requests reconsideration. The cited patent is the U.S. equivalent of WO 00/59515. The rejection should be withdrawn for the same reasons as discussed above in connection with WO 00/59515.

Accordingly, Applicant respectfully requests that the Examiner reconsider and withdraw the rejection of claims 1-7 and 18 under 35 U.S.C. 103(a) as being unpatentable over U.S. Patent 7,026,285 in view of Tang et al.

CONCLUSION

A Notice of Allowance is respectfully requested. The Examiner is requested to call the undersigned at the telephone number listed below if this communication does not place the case in condition for allowance.

If this response is not considered timely filed and if a request for an extension of time is otherwise absent, Applicant hereby requests any necessary extension of time. If there is a fee occasioned by this response, including an extension fee, the Director is hereby authorized to charge any deficiency or credit any overpayment in the fees filed, asserted to be filed or which should have been filed herewith to our Deposit Account No. 23/2825 under docket No.: B0801.70280US01.

Dated: October 20, 2008

Respectfully submitted,

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AMERICAN LUNG ASSOCIATION®

Acute Respiratory Distress Syndrome (ARDS)

What is acute respiratory distress syndrome?

Acute respiratory distress syndrome (ARDS) is the sudden failure of the respiratory system. It can occur in anyone over the age of one who is critically ill. ARDS can be life-threatening because normal gas exchange does not take place due to severe fluid buildup in both lungs.[1] The condition is characterized by rapid breathing, difficulty getting enough air into the lungs and low blood oxygen levels.[2]

Other names for this condition include adult RDS (prior to 1994), increased permeability pulmonary edema and non-cardiac pulmonary edema. Former names include stiff lung, wet lung and shock lung.[3]

Download the Acute Respiratory Distress Syndrome (ARDS) section of Lung Disease Data: 2008.

ARDS is caused mainly by extensive lung inflammation and small blood vessel injury due to sepsis (bacterial infection of the blood), trauma and/or severe pulmonary infection such as pneumonia. However, ARDS also can be linked to multiple transfusions, inhalation of salt water, smoke inhalation of toxic chemicals, aspiration of vomit (inhaling vomit into the lungs), narcotics, sedatives, overdoses of tricyclic antidepressants and shock from any cause.[4]

Onset usually occurs within 24 hours to three days of the original illness or injury.[5]

Cigarette smoking increases the risk of ARDS.[6]

Who has ARDS?

The incidence of ARDS has been difficult to determine partly because of the variety of causes, clinical manifestations and differing criteria used to define it. Various published estimates have ranged from 1.5 to 75 cases per 100,000 persons.[7] In 2007, the National Heart, Lung and Blood Institute estimated that approximately 190,000 Americans are affected by ARDS annually.[8]

What is the health impact of ARDS?

Want to learn more about ARDS and diverse communities? Please view the State of Lung Disease in Diverse Communities: 2007 report.

Approximately 25 percent to 40 percent of ARDS cases are fatal which is an improvement from the ARDS mortality rate of 50 percent to 70 percent just 20 years ago.[9] In 2004, 1,736 deaths due to ARDS were reported.[10] Table 1 displays ARDS incidence and mortality by race. Research shows that men and blacks have higher mortality rates compared to women and other races.[11] Deaths usually result from multi-system organ failure due to the lack of oxygen, rather than lung failure alone. The cause of a patient's ARDS helps predict their chances for survival. The best chances for a positive outcome occur in young trauma-related ARDS patients and patients with fewer chronic health problems. In addition, patients with

milder forms of ARDS tend to have a better chance of recovering than those with a more severe form of the illness. For example, patients who develop ARDS due to sepsis (infection of the blood) usually do not do as well as patients whose ARDS is related to trauma or pulmonary infection.

The greatest risk factors for mortality from ARDS include advanced age, shock and liver failure. Within a week to 10 days, about half of ARDS patients are either deceased or have been weaned off treatment.[12]

Table 1: Acute Respiratory Distress Syndrome Incidence and Mortality [I,II] *

	Incidence		Mortality[3]	
Race	Number[1]	Rate[2]	Number	Rate
Total	190,000	1.5-75.0	1,736	0.6
White	---	---	1,439	0.6
Black	---	---	243	0.8
Hispanic[III]	---	---	54	0.4

Sources

1. National Institutes of Health. National Heart, Lung and Blood Institute. Diseases and Conditions Index. Acute Respiratory Distress Syndrome (ARDS): What Is ARDS? November 2007. Available at http://www.nhlbi.nih.gov/health/dci/Diseases/Ards/Ards_WhatIs.html. Accessed on February 4, 2008.
2. Wheeler AP, Bernard GR. Acute Lung Injury and the Acute Respiratory Distress Syndrome: A Clinical Review. Lancet. 2007; 369:1553-64.
3. Centers for Disease Control and Prevention. National Center for Health Statistics. CDC WONDER On-line Database, compiled from Compressed Mortality File 1999-2004 Series 20 No. 2J, 2007. Accessed on March 4, 2008.

Notes

I. Mortality rates are per 100,000 population and age-adjusted to the 2000 U.S. standard population as of 2004.
II. Acute respiratory distress syndrome incidence (2005) and rates (2003) are per 100,000 population.
III. Hispanics are not mutually exclusive from Whites and Blacks
*** Comparisons should only be made between groups and diseases using rates, not number of cases, as these do not take into account differences which may exist in population size or demographics.**
--- Data not available.

Lung function in most survivors of ARDS will return to normal or near normal within several months; however some will have lasting damage to their lungs or to areas outside the lungs. A study found that one year after discharge from the intensive care unit, ARDS survivors may still suffer side effects, mostly persistent muscle wasting and weakness. Quality of life in these survivors is compromised with poor mental and physical health outcomes.[13]

Want to learn more about ARDS? Please view the fact sheet.

How is ARDS diagnosed and managed?

ARDS is usually diagnosed in a patient who is already critically ill from shock, sepsis or other trauma. The diagnosis is made when there is difficulty in providing adequate oxygenation and diffuse abnormalities on chest x-rays.

Treatment of ARDS involves supportive care in an intensive care unit. Treatment consists of supplemental oxygen and mechanical ventilation along with careful attention to fluid balance and a supportive breathing technique called positive end expiratory pressure (PEEP). These are

co mbined with continuing treatment of the underlying illness or injury.[14]

The goal of mechanical ventilation is to support the patient's breathing during the time needed for the lungs to recover. New advances in mechanical ventilation are being developed. Preliminary results from a study by the National Heart, Lung and Blood Institute suggested that receiving small, rather than large, breaths of air from a mechanical ventilator reduced the number of deaths by 22 percent and increased the number of days without ventilator use.[15]

Pulmonary rehabilitation and support groups are beneficial to survivors.[16]

What is new in ARDS research?

In epidemiological studies, ARDS and acute lung injury (ALI), a less severe form of ARDS, are associated with high mortality rates. Compared to healthy controls and other cases with similar lung disorders, patients with ARDS/ALI had low levels of protein-C and high levels of plasminogen activator inhibitor-1, important proteins in blood clotting. Abnormal levels of these proteins are independently associated with higher mortality and other clinical outcomes such as organ failure. Measuring these levels may provide insight to the development of new therapies.[17] This finding requires further study.

Future research in ARDS will need to focus on mechanistic and cellular studies combined with animal and clinical work, such as determining which animal models translate best to humans and then applying biological marker research to them. Such work will help improve detection and treatment of ARDS.

What is the American Lung Association doing about ARDS?

The American Lung Association is currently funding a number of studies on ALI that could also improve treatment for ARDS. One such study at the University of California, San Francisco is testing a method for improving fluid transport in the lungs, while another at Brigham and Women's Hospital is researching whether altering certain immune system responses will affect inflammation in the lungs.

Want to learn more about how the American Lung Association supports leading research in lung disease? Please view our Research Awards Nationwide listing.

The American Lung Association (www.lungusa.org) sponsors Better Breathers Clubs throughout the country for patients who suffer from chronic lung diseases including chronic obstructive pulmonary disease (chronic bronchitis and emphysema), asthma and others. Also, many hospitals have support groups for people with chronic lung disease.

Thousands of advocates have joined with the American Lung Association to tell Congress that more needs to be done to fight ARDS. Join us to win the battle against lung disease by visiting http://lungaction.org.